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A Lawyer's Medical Guide to Black Lung Litigation

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BLACK LUNG SYMPOSIUM

A LAWYER'S MEDICAL GUIDE TO BLACK LUNG LITIGATION

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INTRODUCTION

The medical and legal professions are easily distinguished from one another by the character of services that each renders to society, yet in many ways, the practitioners of each of these professions "diagnose" and "treat" society's ills. In today's complex society, the two professions find themselves crossing paths more and more often.

Lawyers working in the area of workmen's compensation often become involved both with evidence of a medical nature and also with medical conditions with which they are unfamiliar. Those lawyers who have chosen to devote some or all of their professional time to working in the area of the law dealing with the compensation of the victims of coal workers' pneumoconiosis must have an intimate understanding of its medical aspects.

The purpose of this article is to acquaint attorneys with the medical aspects of coalworkers' pneumoconiosis, or as it is more commonly referred to, black lung. Having this knowledge, the attorney should be in a better position to assist his or her clients in the successful litigation or defense of black lung claims. This article, of necessity, uses many technical medical terms, but in most instances these terms are defined or translated into more common language. In the field of workmen's compensation, the testimony and reports of physicians are essential and the at-

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torney's need to communicate with, examine, or cross-examine physicians, is an integral part of the litigation process. Thus, the attorney needs to be familiar with these technical medical terms.

The article begins in Part I with a brief history of coal miner's lung disease. Part II of the article includes a discussion of the importance of a miner's work history in evaluating the miner's propensity to develop coal workers' pneumoconiosis. In Part III, coalworkers' pneumoconiosis is described in considerable detail including its appearances on chest radiographs, the characteristic pathologic changes in the lungs, its clinical signs and symptoms, its effects on pulmonary function, its effects upon the heart, and its effects upon life expectancy. The last section of the article, Part IV, contains a discussion of several naturally occurring cardiopulmonary diseases including chronic bronchitis, emphysema, asthma, lung cancer, and heart disease. These conditions may produce signs, symptoms, radiographic changes, or pulmonary function changes similar to those elicited by coalworkers' pneumoconiosis (CWP) and therefore often cause diagnostic difficulties.

In addition to the technical description and discussion of coal workers' pneumoconiosis and other cardiopulmonary diseases, the appendix to this article provides a purely practical aid for the black lung lawyer. The appendix delineates ten medically related issues that often arise in the claims litigation process, and summarizes the most probable arguments to be made by both the claimant's lawyer and the defense lawyer.

I. HISTORY OF COAL MINERS' LUNG DISEASES

It has been known for a long time that miners and other workers exposed to silica often developed a chronic, disabling respiratory disease that produced small and sometimes large shadows on the chest radiograph. When it was shown that coal miners often developed similar radiographic shadows, it was assumed that the silica present in the coal dust was responsible for the abnormal chest film. Many Europeans and some Americans still subscribe to this view.[1]

[1] K. MORGAN & A. SEATON, OCCUPATIONAL LUNG DISEASES 149 (1975) [hereinafter referred to as MORGAN & SEATON].

Although coal workers' pneumoconiosis is predominantly associated with underground coal mining, it was the description of a respiratory disease radiographically similar to silicosis, but with a different pathology, occurring among coal trimmers (stevedores) at the Cardiff and Swansea docks in Wales, that clearly separated this disease from silicosis.[2] Because the coal had been washed before it was loaded, and because the loaders had not worked in the mines, it was unlikely that silica could explain the development of their pneumoconiosis. Final support for the hypothesis, and now predominant view among experts, that coal dust or carbon alone might produce pneumoconiosis, came from the demonstration of a similar disease among carbon electrode and electrotype workers.[3]

Within five years of the rediscovery by Gough in 1940 of the occurrence of coal workers' pneumoconiosis in the coal trimmers of Cardiff and Swansea, the Medical Research Council of Great Britain established a Pneumoconiosis Research Unit.[4] Over the next twenty to twenty-five years this Unit contributed significantly to the knowledge of respiratory diseases that occur among coal miners. Americans owe a great deal for their current knowledge about the course and natural history of coal workers' pneumoconiosis to the research conducted by the Pneumoconiosis Research Unit and subsequently to the epidemiologic studies conducted by the National Coal Board.

In the United States, studies of respiratory diseases among coal miners began about 1936. These consisted of epidemiological studies performed with small groups of miners in Pennsylvania,[5] Utah,[6] and also with working bituminous miners from the

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[2] Collis & Gilchrist, *Effects of Dust Upon Coal Trimmers*, 10 J. INDUS. HYGIENE 101 (1928); Gough, *Pneumoconiosis in Coal Trimmers*, 51 J. PATHOLOGY & BACTERIOLOGY 277 (1940).

[3] Watson, Black, Doig, & Nagelschmidt, *Pneumoconiosis in Carbon Electrode Makers*, 16 BRIT. J. INDUS. MED. 274 (1959); Gaensler, Cadigan, Sashara, Foy & MacMahon, *Graphite Pneumoconiosis of Electrotypers*, 41 AM. J. MED. 864 (1966).

[4] MORGAN & SEATON, *supra* note 1.

[5] Dreessen & Jones, *Anthracosilicosis*, 107 J. AM. MED. ASSOC. 1179 (1936).

[6] H. FLINN, R. SEIFERT, H. BRINTON, J. JONES & R. FRANK, SOFT COAL MINER'S HEALTH AND WORKING ENVIRONMENT (Pub. Health Svc. Bull. No. 270, 1956).

tri-state area of western Pennsylvania, Ohio and West Virginia.[7] Some clinical studies were also conducted among groups of anthracite miners from eastern Pennsylvania,[8] bituminous miners from West Virginia[9] and Kentucky.[10] Several other significant studies also occurred during this period.[11] These studies showed that generally, there was a higher incidence of respiratory symptoms and lower ventilatory function among miners, especially those with thirty or more years underground exposure or those over age fifty-five, than among the control groups. The studies also confirmed the findings of earlier British studies[12] which showed that ventilatory function was little influenced by the presence of radiographic opacities of pneumoconiosis until the complicated stages occurred. The American studies also showed that radiographic evidence of pneumoconiosis was positively related to the years spent underground by the miner and further, that motormen more often had large (greater than 3 mm.) nodules on the chest radiograph whereas handloaders more often had small (less than 3 mm.) nodules. Cigarette smoking was also associated with a decline in ventilatory capacity, especially after age fifty.

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[7] Pemberton, *Chronic Bronchitis, Emphysema, and Bronchial Spasm in Bituminous Coal Workers*, ARCH. INDUS. HEALTH 529 (1956) [hereinafter cited as Pemberton].

[8] Motley, Lang, & Gordon, *Pulmonary Emphysema and Ventilation Measurements in One Hundred Anthracite Coal Miners with Respiratory Complaints*, 59 AM. REV. TUBER. 270 (1949).

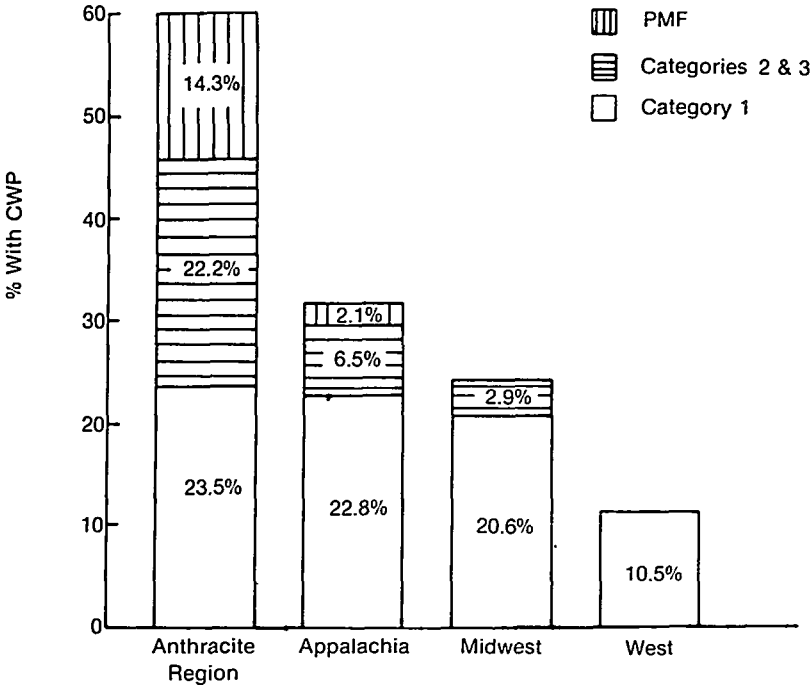
[10] Anderson, Gilbert, & Dossett, *A Comparison of Coal Miners Exposed to Coal Dust and Those Exposed to Silica Dust*, 1 ARCH. ENVT'L HEALTH, 540 (1960).

[11] Hyatt, Kistin, & Mahan, *Respiratory Disease In Southern West Virginia Coal Miners*, 89 AM. REV. RESP. DIS. 387 (1964) (involved a study of a random sample of miners, aged 45-58 years, from Raleigh Co. in southern W. Va.) [hereinafter cited as Hyatt]; Higgins, Higgins, Lockshin, & Canale, *Chronic Respiratory Disease in Mining Communities in Marion County West Virginia*, 25 BRIT. J. INDUS. MED. 165 (1968) (report on an epedemiological study of 425 miners and ex-miners compared to 399 non-miners in five mining communities in northern W. Va.) [hereinafter cited as Higgins]; Rasmussen, Laquer, Futterman, Warren, & Nelson, *Pulmonary Impairment in Southern West Virginia Coal Miners*, 98 AM. REV. RESP. DIS. 658 (1968) (reports on a study of symptomatic miners from southern W. Va., the majority of whom were unemployed and/or seeking compensation) [hereinafter cited as Rasmussen].

[12] J. GILSON & P. HUGH-JONES, LUNG FUNCTION IN COAL WORKERS' PNEUMOCONIOSIS (Med. Research Council Spec. Rep. Ser. No. 290) [hereinafter cited as Gilson].

Two large scale epidemiological studies, one of the Appalachian region only[13] and another of national scope,[14] have

PREVALENCE OF COAL WORKERS' PNEUMOCONIOSIS
IN THE UNITED STATES BY REGION*



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given the best estimates thus far of the incidence of pneumoconiosis, respiratory symptoms and ventilatory function among coal miners.

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[13] W. LAINHART, H. DOYLE, P. ENTERLINE, A. HENSCHEL, & M. KENDRICK, PNEUMOCONIOSIS IN APPALACHIAN BITUMINOUS COAL MINERS, (U.S. Dept of H.E.W., U.S. Govt. Printing Office 1969) [hereinafter cited as LAINHART].

[14] Morgan, Burgess, Jacobson, O'Brien, Pendergrass, Reger, & Shoub, *The Prevalence of Coal Workers' Pneumoconiosis in U.S. Coal Miners*, 27 ARCH. EVNT'L HEALTH 221 (1973).

II. IMPORTANCE OF WORK HISTORY

It is necessary, at the outset of this article, to establish one very basic principle—the functions or jobs a particular miner performs during his tenure in the coal industry is significantly related to that miner's propensity to develop CWP. That being the case, it is important to be aware of both the mining production process in general and the particular work history of the miner whose claim is in issue. It is imperative, in other words, for the black lung attorney to understand the coal mining process as well as the scope, purpose, and work location of various jobs performed in the mining process.

While an attempt to fully outline the various methods of mining coal is beyond the scope of this article, a brief discussion of the various jobs necessary to execute those methods and the relative coal dust exposure of persons performing those jobs (and thus their propensity to develop CWP) is indeed appropriate. While this discussion is in no way exhaustive, it will raise the issue of "work history" in the practitioner's mind and highlight its significance.

A. Coal Mining Methods in the United States

Coal is a complex mineral formed out of decaying organic matter that had become impregnated with a variety of inorganic compounds and subjected to tremendous pressure over millions of years. Coal may be found as surface outcrops or in underground seams, the depths of which vary from a few feet to more than a mile from the surface. Seams that appear as surface outcrops or lie less than about one-hundred feet below the surface can often be mined in an economically feasible manner as an open pit or strip mine.

Prior to about World War II, underground coal mining in the United States was a labor intensive operation which consisted of drilling into the coal face, setting off an explosive, and then, after an appropriate interval, handloading the coal into railroad cars for transport to the surface.[15] Since the late 1940's

[15] Meiklejohn, *History of Lung Disease of Coal Miners in Great Britain: Part I, 1800-1875*, 8 BRIT. J. INDUS. MED. 127 (1951).

larger underground coal mines have become highly mechanized. Two methods of mining are popular. The room and pillar method involves drilling horizontal headers from the vertical shaft to the surface into the coal seam. From these headers, working faces are developed at right angles to the header creating a maze of rooms interspersed by pillars of coal left to support the roof. A machine performs both operations previously consisting of cutting and loading the coal. A roof bolting machine drills into and places long bolts into the overlying rock strata to provide roof support. Electrically powered shuttle cars then transport the coal to the conveyor belts or to underground railroad cars, which move the coal to the surface where it is washed, sized and then stored in silos or dumped via tipples into either railroad cars or barges for transport to its ultimate destination.

The second major method of underground coal mining is known as longwall mining. This, being the usual method of mining in Europe and Britain, was only recently introduced in the United States. Longwall mining involves driving two parallel ingress and egress tunnels with a coal face ranging between fifty and three-hundred yards long between them. A huge "coal plow" is drawn by chains along the coal face "shaving" the coal from the face and causing its descent into a conveyor that moves the loose coal to the transportation system and ultimately to the surface. Large chocks support the roof, providing a safe working place at the face. These chocks are moved forward as the coal face is loaded out, allowing the roof to fall down behind the longwall machine.

Surface or open pit coal mining is accomplished by large capacity shovels and excavators in a non-enclosed space.[16] Thus the exposure of the miner to coal or other dusts that lead to the development of respiratory disease is minimal. The drillers, who drill into the rock strata of the overburden in order to set explosive charges are an exception to this general statement since they may be exposed to silica. Surface miners may also be exposed to coal dust at transfer points such as the conveyor or tipple dumps.

[16] Schlick & Fannick, *Coal in the United States*, in PULMONARY REACTIONS TO COAL DUST 13 (M. Key, L. Kerr, & M. Bundy eds. 1971).

B. Relevance of Work History

All men who work at a coal mine, whether underground or at the surface, are regarded as miners. The areas where the coal is cut is known as the coal face and it is the most dusty part of the mine. The men who operate the cutting machines and continuous miners and their helpers are thus exposed to the highest concentrations of coal mine dust.[17] Shot firers and loading machine operators are only slightly less exposed since their work is also basically at the face. Roofbolters also work just behind the face and are thus similarly exposed, but they also may be exposed to silica from the rock strata above the coal seam. Shuttle car operators who convey the coal from the loading machine to the underground railway and the motorman and brakeman who operate the railway are exposed to smaller amounts of dust than face workers. The motorman and brakeman may, however, be additionally exposed to pulverized silica from sand that is placed on the tracks to obtain traction. Certain miners, whose base of operation is further back from the coal face, are intermittantly exposed to coal dust. These include mechanics, electricians, and general laborers, whose tasks are to maintain the utilities, conveyor belts, tracks and the cage, and also to maintain the machinery and equipment. A few men, known as rock dusters, dust the entrance and haulage ways of the mine with powdered limestone to prevent coal dust explosions. A small number of men work on the surface, either at the tipple or at the portal. Except for time spent working around transfer points of conveyor belts or dumping chutes, these men are not appreciably exposed to coal mine dust.

III. COAL WORKERS' PNEUMOCONIOSIS (CWP)

A pneumoconiosis results from the accumulation of dust in the lung, causing a reaction of the lung tissues.[18] If the inhaled dust contains a large amount of quartz, the condition is called silicosis; if the inhaled dust is coal mine dust, the condition is called coal workers' pneumoconiosis (CWP). In much of the legal literature, CWP is referred to as "black lung," partly as a result

[17] Jacobson, *Respirable Dust in U.S. Bituminous Coal Mines*, in PAPERS AND PROCEEDINGS OF THE NATIONAL CONFERENCE ON MEDICINE AND THE FEDERAL COAL MINE HEALTH AND SAFETY ACT OF 1969, at 68 (1970).

[18] Morgan, Handelsman, Kibelstis, Lapp, & Reger, *Ventilatory Capacity and Lung Volumes of U.S. Coal Miners*, 28 ARCH. ENVT'L HEALTH 182 (1974) [hereinafter cited as Morgan & Handelsman]; MORGAN & SEATON, *supra* note 1.

of the color imparted to the lungs by the deposited dust and partly as a result of the difficulty of pronouncing and spelling the word "pneumoconiosis."[19]

Coal workers' pneumoconiosis (CWP) results from the inhalation of coal mine dust alone. It exists in two forms, simple and complicated (progressive massive fibrosis (PMF)), each identified by the radiographic appearances of the chest film.

Radiology of CWP

A. Simple CWP

Simple CWP is classified into categories 1, 2, and 3 according to the profusion (number per unit area) of small rounded opacities (shadows or nodules) in the lung fields of a radiograph of the chest. When the number of small opacities is insufficient to make a diagnosis of category 1, the film is classified as category 0 or negative ("normal"). The International Labour Organization (ILO) made the first attempt to codify the interpretations of chest radiographs for pneumoconiosis in 1958.[20] This was followed by a scheme devising a twelve-point elaboration of the ILO classification system that rendered it more sensitive for the purpose of reading radiographic progression.[21] In this latter study, each major category, including zero, was divided into three subcategories, so that in the full elaboration there were 12 categories ranging from 0/- to 3/4.

RELATIONSHIPS BETWEEN PROFUSION ON THE ELABORATED ILO U/C AND THE SHORT FORM CLINICAL CLASSIFICATION OF THE RADIOGRAPHIC APPEARANCES OF THE PNEUMOCONIOSES

0/- 0/0 0/1	1/0 1/1 1/2	2/1 2/2 2/3	3/2 3/3 3/4
Category 0	Category 1	Category 2	Category 3
No Pneumoconiosis	Definite Pneumoconiosis		

[19] Strader & Sheche, *Federal Black Lung: Ten Years of Legislation and Litigation*, Presented to ABA Annual Meeting (1980).

[20] INTERNATIONAL LABOUR ORGANIZATION, MEETINGS OF EXPERTS ON THE INTERNATIONAL CLASSIFICATION OF RADIOGRAPHS OF THE PNEUMOCONIOSES, OCCUP. SAFETY & HEALTH SERIES 63 (1959).

[21] Liddell & May, *Assessing The Radiological Progression of Simple Pneumoconiosis*, NAT'L COAL BOARD MED. SER., (1966).

For example, when a radiograph is being classified and the reader initially considers category 1, but eventually decides there are too few opacities, causing the correct classification to be category 0, then the classification of that radiograph is 0/1. The same applies to categories 1/2, 2/1, 2/3, 3/2 and 3/4. The numerator represents the category in which the film is placed, and the denominator represents the category that was also considered. If the interpretor does not consider any other category but the one in which he places it, then the film is classified as 1/1, 2/2, or 3/3.

Small opacities are also classified as to whether they are regular (rounded) or irregular (linear, reticular). The regular or rounded opacities are primarily seen on the radiographs of coal miners. The regular opacities are classified according to size, pin-

SIZE AND SHAPE DESIGNATIONS OF ROUNDED AND IRREGULAR OPACITIES ON THE ILO U/C CLASSIFICATION OF THE RADIOGRAPHIC APPEARANCES OF THE PNEUMOCONIOSES

Rounded Opacities		Irregular Opacities	
p	1.5 mm	s	fine
q	1.5 to 3.0 mm	t	medium
r	3.0 to 10.0 mm	u	coarse

head (p), ranging up to 1.5 mm. in diameter, micronodular (q), ranging from 1.5 to 3.0 mm. in diameter and nodular (r) ranging from 3.0 to 10.0 mm. in diameter. Irregular opacities are commonly seen in asbestosis and certain other interstitial reactions but are infrequent among coal miners. When present among coal miners, these irregular opacities relate more to cigarette smoking than to dust retention.[22] The irregular opacities are also classified by size into (s), up to 1.5 mm., (t) between 1.5 and 3.0

[22] Amandus, Lapp, Jacobson, & Reger, *Significance of Irregular Small Opacities in Radiographs of Coal Miners in the U.S.A.*, 33 BRIT. J. INDUS. MED. 13 (1976).

mm., and (u) between 3.0 and 10.0 mm. in width, according to the latest classification scheme.[23]

B. Complicated CWP

Complicated pneumoconiosis or progressive massive fibrosis (PMF) is diagnosed when any opacity reaches a size of 1 cm. or more in diameter. It is classified as category A, B, or C according

SIZE DESIGNATIONS OF THE LARGE OPACITIES IN COMPLICATED PNEUMOCONIOSIS

Large Opacities
A - 1.0 to 5.0 cm
B - >5.0 cm but < RUZ
C - > RUZ (1/3 of one lung)

to the size of the large opacity or opacities. When an opacity or opacities whose combined diameter is between 1 and 5 cm. is seen on the chest radiograph, it is classified as category A. A chest radiograph showing an opacity or opacities having a combined diameter of greater than 5 cm., but less than one-third of one lung, is classified as category B. If the opacities occupy more than one third of one lung, the chest radiograph should be classified as category C. Complicated CWP or PMF almost always develops on a background of category 2 or 3 simple pneumoconiosis. Therefore the presence of a large opacity on a background of category 1 or less should be viewed as a lung tumor, or due to a disease other than complicated CWP.

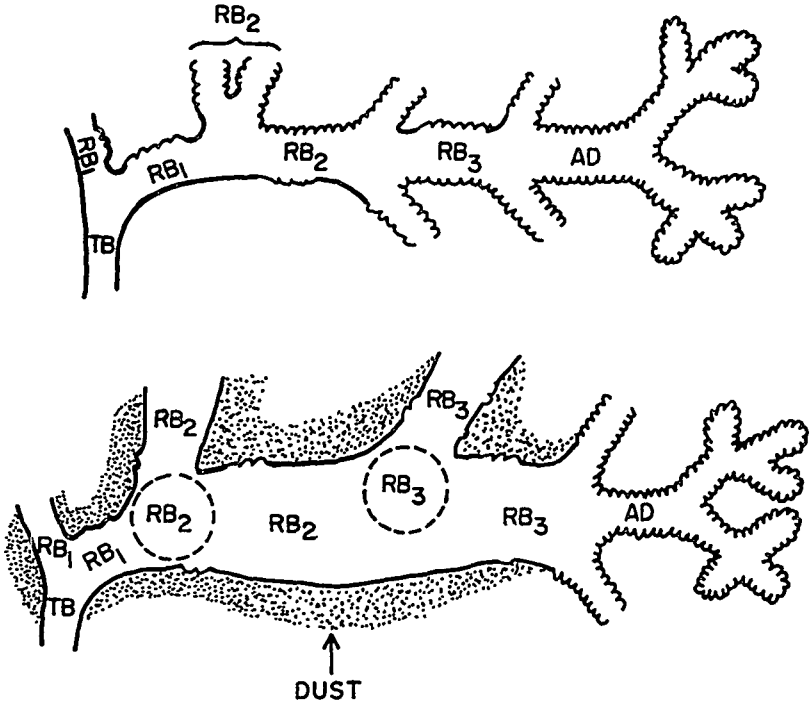
[23] INTERNATIONAL LABOUR OFFICE, GUIDELINES FOR USE OF ILO INTERNATIONAL CLASSIFICATION OF RADIOGRAPHICS OF PNEUMOCONIOSIS (Occup. Safety and Health Ser. No. 22) (rev. ed. 1980).

Pathology of CWP

A. Simple CWP

Failure of, or extreme interference with the normally efficient mechanisms for clearance of dust and foreign material from the parenchymal (alveolar, gas exchanging) parts of the lungs results in the characteristic lesions of simple CWP. Coal dust which is deposited in the alveoli is either phagocytosed (ingested) by macrophages (scavenger cells) and conveyed centrally toward the larger airways where these cells, containing dust particles, mount and ride a mucous escalator to the mouth or they enter lymph channels and are transported to lymph nodes

REPRESENTATION OF THE NORMAL ANATOMY OF THE TERMINAL LUNG UNIT IN THE DEVELOPMENT OF THE CHARACTERISTIC LESIONS OF CWP, THE COAL MACULE, WITH ITS ASSOCIATED FOCAL EMPHYSEMA*



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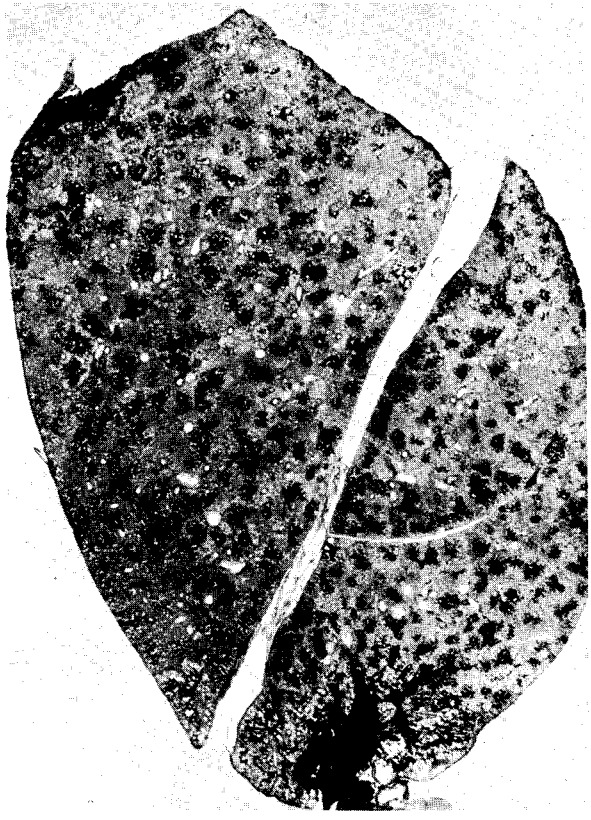
(filtering stations) at the root of the lung. Those particles which are ingested by the scavenger cells and ultimately exit the mouth in the form of mucous do not contribute to the occurrence of CWP. Neither do those particles which are transported to the lymph nodes at the root of the lung contribute to the development of CWP, however their presence is indicative of coal dust exposure.

In time, small accumulations of dust begin to aggregate around the respiratory bronchiole (small airway leading into the alveolar region). When the dust-laden macrophages become so numerous that they can no longer pass beyond the respiratory bronchiole, they accumulate and build up a mantle (collar) of coal

PAPER MOUNTED SECTION OF AN INFLATED LUNG DEMONSTRATING PRIMARILY SIMPLE CWP*



*An early complicated lesion is seen at the top of the section.

dust. The mantle weakens the wall of the respiratory bronchiole allowing it to dilate (enlarge its opening). The combination of the stellate (star-shaped) mantle of coal dust and the associated dilatation of the respiratory bronchiole is the characteristic lesion of CWP. These formations and their microscopic appearances have been carefully described;[24] they may get as large as 5 mm. in diameter and often are more numerous in the upper lobes of the lungs. These formations are best visualized, at an autopsy, on lungs that are fixed in the inflated position, sliced thinly from top to bottom and viewed either with low magnification or mounted on white paper.

Miners who are exposed to quartz as well as coal dust develop lesions that have features of both CWP, as described above, and classical silicosis. These are best regarded as mixed dust lesions, and the mantles, formed as a result of dust accumulation, tend to be round rather than the stellate (star-shaped) macules of simple CWP. They contain more collagen (scar tissue), and are often arranged in concentric layers, interspersed with dust. Special techniques (polarized light for example) demonstrate the presence of silica and silicates in these micro and macro nodules. The nodules range in size from somewhat less than 7 mm. to nearly 1 cm. in diameter, have a grey-black appearance, and are found in the center of the secondary lobule, that is, either around, or incorporating, the respiratory bronchiole.[25]

B. Complicated CWP

The pathological lesions of complicated CWP (PMF) first appear in the posterior segment of one or the other upper lobe, or in the superior segment of the lower lobe.[26] They consist of large collections of ill-defined black tissue which are rubbery in texture and often adhered to the chest wall. The lesions may demonstrate cavity formation in their center.[27] Viewed micro-

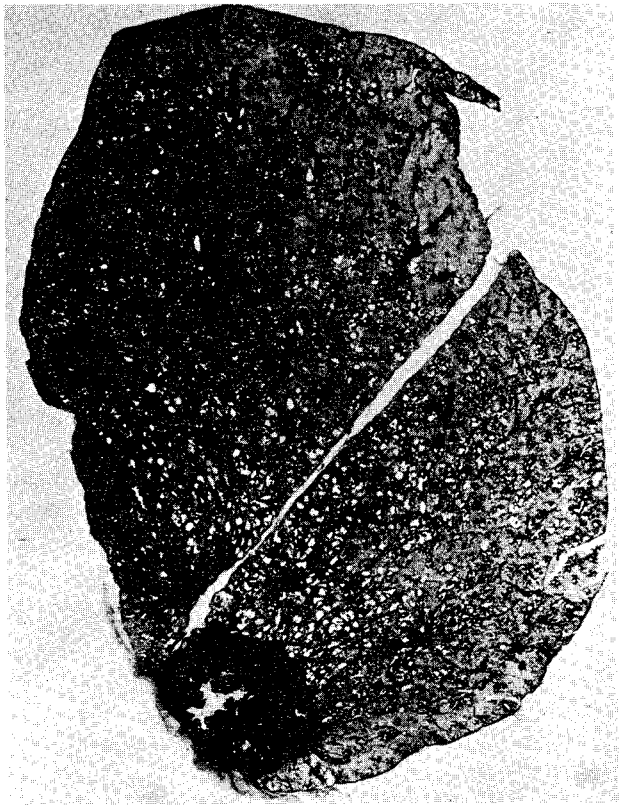
[24] Heppleston, *The Essential Lesion of Pneumokoniosis In Welsh Coal Workers*, 59 J. PATHOLOGY & BACTERIOLOGY 453 (1947).

[25] Kleinerman, Green, Laquer, Taylor, Harley, Pratt, Wyatt, Naeye, Wiot, & Lapp, *Pathology Standards For Coal Workers' Pneumoconiosis*, 103 ARCH. PATH. & LAB. MED. 375 (1979) [hereinafter cited as Kleinerman].

[26] James, *The Relationship of Tuberculosis to the Development of Massive Pneumoconiosis In Coal Workers*, 48 BRIT. J. TUBERC. 89 (1954) [hereinafter cited as James].

[27] Kilpatrick, Peppleston, & Fletcher, *Cavitation in Massive Fibrosis of Coal Worker's Pneumoconiosis*, 9 THORAX 260 (1954).

PAPER MOUNTED SECTION OF INFLATED LUNG DEMONSTRATING EXTENSIVE BACKGROUND OF SIMPLE CWP UPON WHICH IS SUPERIMPOSED LARGE CONGLOMERATE MASSES OF COMPLICATED PNEUMOCONIOSIS IN THE UPPER ZONE



scopically, the lesions of complicated CWP (PMF) appear to be composed of a capsule of thick scar tissue surrounding a formless black mass. Traces of obliterated arteries, veins, and bronchioles that were incorporated into and destroyed by the process may occasionally be seen within the formless masses.[28]

Complicated CWP (PMF) always occurs on a background of simple CWP. While some researchers have chosen to use an ar-

[28] Wells, *Pulmonary Vascular Changes in Coal Worker's Pneumoconiosis*, 68 J. PATHOLOGY & BACTERIOLOGY 573 (1954).

bitrary size of 3 cm. or greater in defining PMF,[29] others have similarly elected to use 1 cm. or greater. Recognizing that solitary nodules are generally larger on autopsy examination than they appear on a chest radiograph, the Pneumoconiosis Committee of the College of American Pathologists[30] recommended that a nodule be at least 2 cm. or larger on autopsy to define the presence of complicated CWP or PMF.

Clinical Signs and Symptoms

A. Simple CWP

Simple CWP seldom elicits symptoms or signs of abnormality. The usual course is for the nodular opacities to develop silently over the course of many years. Attention is called to these nodules when some intercurrent problem arises that leads to the making of a chest radiograph or the film is made as part of a monitoring or surveillance program. Although many miners with CWP complain of cough and sputum, these symptoms are seen with equal frequency in miners with clear chest films. These common symptoms may be attributed in part to a type of occupationally related chronic bronchitis. The additional presence of severe shortness of breath as well is virtually always a consequence of non-occupationally related disease such as chronic bronchitis or emphysema.

Subjects with category B and C complicated CWP usually complain of excessive dyspnea (shortness of breath) on exertion. They often cough and produce sputum which, often because of the rupture of a large cavitary lesion into the airway, will appear as an inky black material. The production and appearance of this inky black material is known as melanoptysis and is the only truly characteristic symptom of CWP. In contrast to cancer, with which large PMF lesions may be confused, symptoms involving chest pain and hemoptysis (coughing up of blood) in the sputum are rarely observed in CWP victims.

The signs of complicated CWP are those of consolidation and collapse (dullness, lack of air entry) of the affected area of the lung. Clubbing (abnormal accumulation of tissue and softening)

[29] James, *supra* note 26.

[30] Kleinerman, *supra* note 25.

of the fingers is not seen.[31] Advanced complicated CWP may result in the development of pulmonary arterial hypertension (increased pulmonic component of second heart sound), right ventricular failure (swelling of feet, enlarged liver, fluid accumulation in abdomen) and cor pulmonale (right ventricular disease and failure secondary to lung disease).

Complicated CWP is not invariably progressive, but unlike simple CWP, it usually advances slowly over the course of five to fifteen years, assuming the absence of further dust exposure. Of those miners with category 2 or 3 simple CWP, 1.5 to 2 percent per year will ultimately develop complicated CWP.[32] Thus, complicated CWP is likely to develop in about fifty percent of the men between the ages of forty and seventy who are affected by category 2 or 3 simple pneumoconiosis.[33]

Pulmonary Function in CWP

The extent and nature of cardiopulmonary (heart and lung) functional impairment found among coal miners depends, to a large extent, on subject selection. Random selection of working miners yields a sample of mostly asymptomatic, physically fit subjects. When non-working miners are studied, the sample contains a significant number of the less fit, i.e., those who left the industry because of poor health. It is desirable to study both working and non-working groups to obtain a true picture of the functional impairments that are attributable to the industry. Very few such studies have been made so it is necessary to rely on studies of one or the other group for our data on the cardiopulmonary effects of CWP and exposure to coal mine dust.

Ventilatory Function

Ventilatory function refers to the capacity of the lungs to accept, and the integrity of the airways which conduct, oxygen into and the waste gas, carbon dioxide, out of the gas exchange units. This is sometimes referred to as the bellows function of the

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[31] Morgan & Lapp, *State of the Art: Respiratory Disease in Coal Miners*, 113 AM. REV. RESP. DIS. 531 (1976) [hereinafter cited as Morgan & Lapp].

[32] *Id.*

[33] Leathart, *Clinical Aspects of Respiratory Disease Due to Mining*, in MEDICINE IN THE MINING INDUSTRY 83 (J. Rogan ed. 1972).

lungs. Ventilatory function is measured by the technique of spirometry and is quantified as the Forced Vital Capacity (FVC) and its subdivisions of timed lung volumes and flow rates. From a recording of the FVC maneuver one can derive (1) the volume of air exhaled in the first second ($FEV_{1.0}$), (2) the forced expiratory flow between 200 and 1200 ml. exhaled ($FEF_{200\text{-}1200}$) which is approximately equal to maximal or peak flow, and (3) the average flow over the middle half of the maneuver ($FEF_{25\text{-}75\%}$). These tests are all used to detect and quantify air flow obstruction, thus assisting in the diagnosis of diseases primarily involving the airways.

Several attempts have been made to establish the effect of coal mining on ventilatory function. One study found that the mean $FEV_{1.0}$ of the miners was slightly lower than either of two age-matched control groups.[34] Using $FEF_{25\text{-}75\%}$ as its index of lung function, another study[35] found an association between increasing ventilatory impairment and both the prevalence of bronchitis and years worked in underground mines. Only category 3 simple CWP and complicated CWP were associated with a decreased $FEF_{25\text{-}75\%}$. A further study,[36] using the $FEV_{1.0}$ to assess functional status, found that miners and ex-miners with more than thirty years of underground exposure had a statistically significantly lower $FEV_{1.0}$ than non-miners from the same communities. There was, however, no significant difference between non-miners and miners with less than thirty years underground exposure.

A random sample of nearly thirty-five hundred working and non-working miners from the entire Appalachian region were studied by the Public Health Service.[37] Both bronchitis and cigarette smoking were associated with a decrease in $FEV_{1.0}$, but no relationship was shown between a decrease in $FEV_{1.0}$ and the radiographic category of CWP except in instances of complicated CWP. The National Coal Study[38] involved a random sample of more than nine-thousand working miners or approximately ten percent of all working miners throughout the United

[34] Pemberton, *supra* note 7.
[35] Hyatt, *supra* note 11.
[36] Higgins, *supra* note 11.
[37] LAINHART, *supra* note 13.
[38] Morgan & Handelsman, *supra* note 18.

States. This study, demonstrating a slightly lower than predicted $FEV_{1.0}$, elicited no evidence of a clear relationship between simple CWP and reduced $FEV_{1.0}$, however, in all regions the presence of complicated CWP was associated with significant decreases in $FEV_{1.0}$.

In all of the studies cited, respiratory symptoms were associated with a decrease in ventilatory function and cigarette smoking was by far the most important factor in producing respiratory symptoms. The contributing effect of simple CWP and years spent in underground mines upon a decrease in ventilatory function are relatively slight by comparison to smoking. Studies comparable to those discussed above have been carried out in Great Britian[39] and in Germany[40] with essentially the same findings.

Lung Volumes

Lung volumes refer to the amount of gas present in the lungs when an individual inhales fully (total lung capacity, TLC) and when the individual exhales fully (residual volume, RV). Normal TLC (total lung capacity) decreases when some disease process in the lungs (pulmonary) or disease involving the chest wall or linings of the lungs (extrapulmonary) causes a restrictive disorder. RV (residual volume) increases abnormally when there is air trapping, a condition most often caused by premature closure of the small airways leading into the gas exchange units. Emphysema (destruction of alveolar walls) or any disease of the airways will usually precipitate this air trapping phenomenon.

While one study[41] was unable to detect changes in the lung volumes that were related to the presence of simple CWP in

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[39] Cochrane & Higgins, *Pulmonary Ventilatory Functions of Coalminers in Various Areas in relation to the X-ray Category of Pneumoconiosis*, 15 BRIT. J. PREVENTIVE SOC. MED. 1 (1961); Ashford, Brown, Morgan, & Rae, *The Pulmonary Ventilatory Function of Coal Miners in the United Kingdom*, 97 AM. REV. RESP. DIS. 810 (1968) [hereinafter cited as Ashford]; Higgins & Oldham, *Ventilatory Capacity in Miners*, 19 BRIT. J. INDUS. MED. 65 (1962); Muir, *Pulmonary Function in Miners Working in British Collieries: Epidemiological Investigations by the National Coal Board*, 11 BULL. PHYSIOPATH. RESP. 403 (1975).

[40] Ulmer & Reichel, *Functional Impairment in Coal Workers' Pneumoconiosis*, 200 ANN. NEW YORK ACAD. SCI. 405 (1972).

[41] Gilson, *supra* note 12.

British coal miners, another,[42] using a radiographic method of determining lung volumes in nearly fifteen-hundred working Pennsylvania coal miners, found a slight increase in TLC which was related to increasingly serious categories of simple CWP. In this latter study, a much more striking increase in the observed RV, as a ratio of the predicted value, was found. This increase in the RV was related to increasingly serious categories of simple CWP in an almost linear manner. An increase in RV alone could be due to intrinsic disease of the airways or to emphysema (destruction of alveolar walls). The fact that both RV and TLC were increased suggests that emphysema was the most significant factor contributing to the result. Furthermore, the fact that non-smoking miners who were without measurable airway obstruction showed this increase in RV and also the fact that this change was related to radiographic simple CWP implicates the focal emphysema, that occurs in relation to the coal mantle, as the cause for the air trappings.

Diffusing Capacity

The carbon monoxide diffusing capacity of the lung (D_{LCO}) is a test for overall gas exchange that does not require sampling of arterial blood. Factors that influence this test are (1) the surface area (volume) of the alveoli, (2) the adequacy of the pulmonary capillary blood volume, (3) the uniformity of matching and the thickness of the membranes separating the alveoli from the capillaries (blood vessels), (4) the amount of hemoglobin in the red blood cells in the capillaries, and (5) the ability of the hemoglobin to chemically combine with the carbon monoxide gas molecules. In conjunction with other tests, such as spirometry and those designed to measure lung volumes, this test can sensitively detect, during life, the presence of emphysema (destruction of alveolar walls with their associated capillary vessels) and diseases involving thickening, scarring or inflammation of the alveolar walls.

Studies using as their basis the diffusing capacity of the lung (D_{LCO}) have been made among miners in Europe, Great Britain and the United States.[43] These may be summarized by say-

[42] Morgan, Burgess, Lapp, Seaton, & Reger, *Hyperinflation of Lungs in Coal Miners*, 26 THORAX 585 (1971); Morgan, Seaton, Burgess, Lapp, & Reger, *Lung Volumes in Working Coal Miners*, 200 ANN. NEW YORK ACAD. SCI. 478 (1972).

[43] Morgan & Lapp, *supra* note 31, at 539.

ing that in tests among miners suffering from simple CWP there is little deviation from the norm shown by this test of gas exchange. Miners with the p size opacities did have a statistically significant lower value than other types of simple CWP, but even these values were not outside the normal range. In contrast, miners with advanced categories of complicated CWP, those categorized in the B or C group, had stikingly abnormal values for D_{LCO}.[44]

Gas Exchange

Gas exchange may also be measured directly by a simultaneously sampling a subject's arterial blood and collecting their exhaled air both at rest and during exercise. These samples are then analyzed both for partial pressures or concentrations of oxygen and carbon dioxide, noting particularly the volume of gas collected within a measured period of time. Studies involving these measurements among non-smoking or unobstructed miners demonstrate nearly normal oxygen partial pressure (Pa_{02}) among those suffering from simple CWP.[45] The miner achieves this normal oxygenation only as a result of some hyperventilation. Hyperventilation overcomes the slight mismatching between ventilation and blood flow, which manifests itself as an abnormal widening of the alveolar-arterial oxygen difference $(A\text{-}a)_{02}$. Similar findings among miners with simple CWP have been observed in studies conducted in Great Britain[46] and Belgium.[47] Miners with advanced categories of complicated CWP, those categorized in the B or C group, who were studied in the same manner described above, showed normal oxygenation of the blood while at rest, however their oxygenation level became significantly reduced during modest exercise.[48]

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[44] Lapp & Seaton, *Pulmonary Function*, in PULMONARY REACTIONS TO COAL DUST: A REVIEW OF UNITED STATES EXPERIENCE 153 (M. Key, L. Kerr, & M. Bundy eds. 1971) [hereinafter cited as Lapp & Seaton].

[45] *Id.*

[46] Cotes, Deivanayagam, Field, & Billiet, *Relationship Between Type of Simple Pneumoconiosis (p or m) and Lung Function*, in 2 INHALED PARTICLES III 633 (W. Walton ed. 1971).

[47] Frans, Veriter, & Brasseur, *Pulmonary Diffusing Capacity for Carbon Monoxide in Simple Coal Workers' Pneumoconiosis*, 11 BULL. PHYSIOPATH. RESP. 479 (1975).

[48] Lapp & Seaton, *supra* note 44.

Hemodynamics

Several studies involving Appalachian miners who were either disabled or seeking compensation seemed to demonstrate that pulmonary hypertension (elevation of pressure within pulmonary blood vessels) was fairly common among those having worked in the mines.[49] This is in contrast to European studies[50] which showed (1) that pulmonary hypertension was uncommon in unobstructed miners with simple pneumoconiosis, (2) that when pulmonary hypertension occurred it was a consequence of bronchitis, and (3) that bronchitic miners with pulmonary hypertension did not differ from non-miners with bronchitis. A further study,[51] involving obstructed and non-obstructed Appalachian coal miners, demonstrated findings that were consistent with the European studies, namely that (1) pulmonary hypertension was uncommon among unobstructed miners with simple CWP and (2) that miners with advanced categories of complicated CWP and also those suffering from obstructive airway disease more commonly demonstrate pulmonary hypertension.

Further investigations of the pulmonary vascular bed were performed in a study using the techniques of radioactive tagged lung scans,[52] a technique permitting one to study the integrity of those smaller blood vessels of the lungs whose inner diameters are less than about 50 μm (1/500th of an inch). This study demonstrated that complicated CWP is regularly associated with defects in perfusion (blood supply) in the areas around the large

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[49] Rasmussen, Laquer, Futterman, Warren, & Nelson, *Pulmonary Impairment in Southern West Virginia Coal Miners*, 98 AM. REV. RESP. DIS. 658 (1968); Stoeckle, Hardy, King, & Nemiah, *Respiratory Disease in U.S. Soft Coal Miners: Clinical and Etiological Considerations*, 15 J. CHRONIC DIS. 887 (1962).

[50] Cotes, *In Discussion*, 4 BULL. PHYSIOPATH. RESP. 207 (1968); Kremer & Lavenne, *La Circulation Pulmonaire dans les Pneumoconioses des Houilleurs*, 22 POUMON. COEUR 767 (1966); Kremer, Timmerman, Baudrez, & Lambrecht, *Hemodynamique Pulmonaire dans les Pneumoconioses des Houilleurs*, 22 REV. INST. HYGEINE MINES 3 (1967); Navratil, Widimisky, & Kasalicky, *Relationships of Pulmonary Hemodeynamics and Ventilation and Distribution in Silicosis*, 4 BULL. PHYSIOPATH. RESP. 349 (1969).

[51] Lapp, Seaton, Kaplan, Hunsaker, & Morgan, *Pulmonary Hemodynamics in Symptomatic Coal Miners*, 105 AM. REV. RESP. DISEASE 418 (1971).

[52] Seaton, Lapp, & Chang, *Lung Perfusion Scanning in Coal Workers' Pneumoconiosis*, 103 AM. REV. RESP. DIS. 338 (1971).

nodules and also in the areas of emphysematous bullae (holes) adjacent to the nodules, which are caused by the overstretching of the lung by contracting scar tissue. The study also showed that the vascular bed is normal in most subjects with simple CWP. These findings among living miners correspond very closely with findings at the autopsy stage, by others,[53] who described cor pulmonale (pulmonary heart disease) as present among some with complicated CWP, but seldom, if ever, among those with simple CWP.

Further evidence that simple CWP is not associated with cor pulmonale (pulmonary heart disease) is derived from another study which involved a group of twenty-four miners with simple CWP and an age matched control group.[54] The researchers used vectorcardiograms, which are special three-dimensional electrocardiograms. This technique is especially sensitive to enlargement of the right ventricle or to rotational changes of the heart that are recognized as early signs of cor pulmonale. The study showed no differences between the vectorcardiograms of the miners with simple CWP and those in the control group. Since the miners with CWP in this study were also one of the groups that underwent cardiac catheterization with direct measurement of pulmonary vascular pressures, it is not possible that the vectorcardiograms were missing any pulmonary heart disease.

Lung Mechanics

Pulmonary mechanics refers to the measurement of pressure, airflow and volume relationships simultaneously and in relation to each other. Alterations in these indices in one direction indicate abnormal stiffness of the lungs caused by a build up of scar tissue. Alterations in the opposite direction indicate destruction of the normal lung tissue which characteristically occurs in emphysema. These kinds of measurements have been made among coal miners in an attempt to characterize the mechanical alterations, if any, associated with simple and complicated CWP.

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[53] Wells, *supra* note 28; James & Thomas, *Cardiac Hypertrophy in Coalworkers' Pneumoconiosis*, 113 BRIT. J. INDUS. MED. 24 (1956).

[54] Hunsaker, Kaplan, Donnelly, & Lapp, *The Frank Vectorcardiogram in Coal Miners' Pneumoconiosis*, 3 J. ELECTROCARDIOLOGY 155 (1970).

The findings from the studies of lung mechanics in coal miners may be summarized in the following statements. Miners with simple CWP showed minor alterations in the static pressure-volume behavior of the lungs, consistent with mild or focal emphysema. These measurements among miners with complicated CWP were consistent with fibrosis or scarring.[55] As compared with normal standards of lung function, the measurement of these indices under conditions of rapid breathing and among miners with simple CWP and no detectable abnormalities, by usual test of lung function, did reveal evidence of some obstruction in small airways leading into the gas exchange parts of the lungs.[56] Further demonstration of this phenomenon, among miners with simple CWP, that is premature closure of small airways, by the use of another technique, confirmed the findings suggested by the earlier mechanics.[57]

IV. RELATIONSHIP OF COAL MINING TO OTHER CARDIOPULMONARY DISEASES

The occurrence of symptoms of cough, sputum, shortness of breath, wheezing, pain in the chest or weight loss in a coal miner cannot automatically be assumed to be arising from CWP or necessarily a consequence of coal mining exposure. Coal miners, like other individuals, smoke cigarettes, inherit the predisposition to develop allergies and asthma, are exposed to air pollution, and may contract respiratory infections, develop hypertension, coronary artery disease or lung cancer. Since the heart and lungs have relatively few, stereotyped ways of manifesting symptoms that are due to a wide variety of causes, diseases of diverse origins may be present with the same cardiac or pulmonary symptoms. By careful inquiry and physical examination, it is usually possible to sort out the various causes for these common symptoms. It it therefore necessary to briefly discuss what, if any, relationship there is between naturally occurring condi-

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[55] Seaton, Lapp, & Morgan, *Lung Mechanics and Frequency Dependence of Compliance in Coal Miners*, 51 J. CLIN. INVEST. 1203 (1972).

[56] *Id.*; Lapp & Seaton, *Lung Mechanics in Coal Workers' Pneumoconiosis*, 200 ANN. NEW YORK ACAD. SCI. 433 (1972); Morgan, Lapp, & Morgan, *The Early Detection of Occupational Lung Disease*, 68 BRIT. J. DIS. CHEST 75 (1974).

[57] Lapp, Block, Buchleck, Lippmann, Morgan, & Reger, *Closing Volume in Coal Miners*, 113 AM. REV. RESP. DIS. 155 (1976).

tions that produce the same symptoms as CWP and an individual's occupational exposure in coal mining.

Industrial Bronchitis

Chronic bronchitis is a respiratory disease defined in clinical terms by the American Thoracic Society[58] as a condition characterized by the presence of cough and phlegm on most days for at least three months of the year, for at least two consecutive years, and not due to localized disease such as tuberculosis, pneumonia, cancer, or bronchiectasis. It is characterized pathologically by hypertrophy (enlargement) and hyperplasia (increased number) of bronchial mucous glands and increased numbers of goblet cells in the mucosa (lining) of the small airways. In chronic bronchitis, the inner channel of the airways (lumen) is frequently narrowed by mucus secretion.[59] Epidemiological studies have shown that often the most important single factor associated with sputum production and increases in the mucus-secreting glands and goblet cells is tobacco smoking. While other conditions such as climate and industrial exposure may also contribute, the contributions of these may not be detectable unless only non-smokers are studied.[60]

Thus the Medical Research Council of Great Britain concluded in 1966, with particular reference to the coal mining industry, that "on present evidence, intensity of dust exposure does not appear to be a very significant factor in determining the prevalence of bronchitis in this group of workers."[61] However, several studies involving large populations of working coal miners have reported an increase in the symptoms of bronchitis among the miners, thus suggesting that increased dust exposure may have been a contributing factor. These studies, however, have relied upon indirect measures of dust exposure such as chest radiographs or years worked underground by the miner,[62]

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[58] W. THURLBECK, CHRONIC AIR FLOW OBSTRUCTION IN LUNG DISEASE (1976).

[59] *Id.*

[60] Reid, *The Pathology of Coal Pneumoconiosis*, in MEDICINE IN THE MINING INDUSTRIES 1 (J. Rogan ed. 1972).

[61] Medical Research Council, *Chronic Bronchitis and Occupation*, 1 BRIT. MED. J. 101 (1966).

[62] Ashford, *supra* note 39; Rogan, Ashford, Chapman, Duffield, Fay, & Rae, *Pneumoconiosis and Respiratory Symptoms in Miners at Eight Collieries*, BRIT.

and thus the reliability of the studies' conclusions are somewhat in question.

Direct confirmation of the effect of airborne dust in coal mining on increasing the prevalence of bronchitis in British coal miners was provided by one study[63] which was able to calculate lifetime exposures based upon measurements of respirable dust over a ten-year period. This study found a signficant relationship between dust exposure and the prevalence of bronchitis in those aged twenty-five to thirty-four and thirty-five to forty-four years. It also found that in all age groups, cigarette smoking and the presence of CWP were associated with an increased prevalence of bronchitis.

Using flow volume curves of forced expiration, derived from tests conducted among working American coal miners, a recent study[64] was able to show that smoking was associated with reductions in airflow at all lung volumes. However, non-smoking miners who had symptoms of bronchitis demonstrated only reductions in peak flow and flows at the higher lung volumes when compared to non-smoking miners without bronchitis. There is strong evidence that flows at the higher lung volumes reflect the state of the larger airways where larger particles would tend to be deposited and cleared out without causing pneumoconiosis. It thus seems established that the chronic inhalation of dust is capable of inducing chronic bronchitis, which might rightfully be labeled industrial or occupational, in accordance with its origin. Further, this bronchitis may occur in the absence of pneumoconiosis (clear chest film) because the particles are larger than those that penetrate into the alveoli and produce the typical radiographic picture.

Industrial bronchitis is not restricted to those in the coal mining industry; it has also been found among steel workers,[65]

MED. J. 1337 (1961); Kibelstis, Morgan, Reger, Lapp, Seaton, & Morgan, *Prevalence of Bronchitis and Airway Obstruction in American Bituminous Coal Miners*, 108 AM. REV. RESP. DIS. 886 (1973) [hereinafter cited as Kibelstis].

[63] Rae, Walker, & Altfield, *Chronic Bronchitis and Dust Exposure in British Coal Miners*, in 2 INHALED PARTICLES III 883 (W. H. Walton ed. 1971).

[64] Hankinson, Reger, Fairman, Lapp, & Morgan, *Factors Influencing Expiratory Flow Rates in Coal Miners*, in 2 INHALED PARTICLES IV 737 (W.H. Walton ed. 1977).

[65] Lowe, Campbell, & Khosla, *Bronchitis in Two Integrated Steelworks*, 27 BRIT. J. INDUS. MED. 121 (1970).

textile workers,[66] gold miners,[67] and cement workers.[68] Industrial bronchitis appears to be a nonspecific reaction in the airways of any group of workers exposed to dust particles. The important points are (1) that industrial bronchitis has an insignificant clinically effect upon ventilatory capacity[69] and (2) that the effects of smoking upon ventilatory function are significant, causing impairment to be from two to ten times greater among smokers than among non-smokers, depending upon which index of ventilatory function is chosen as the standard of measurement.

Emphysema

Emphysema is defined as the enlargement of the air spaces distal to the terminal bronchiole produced by destruction of alveolar walls (septae).[70] The cause of emphysema is unknown, but many researchers think that the primary mechanism is damage to the blood capillaries traversing the alveolar wall. The reduction or closing off of the blood supply weakens the wall which in turn ruptures under the normal stress of inflating the lung and this in turn leads to an abnormal enlargement of the air spaces and the alteration in lung distensibility that is characteristic of emphysema.

Emphysema occurs in several different forms, each of which is identified by the location of the enlarged air spaces within the acinus (terminal lung unit).

Focal emphysema is a specific lesion that occurs in and around coal mantles (dust aggregates). The presence in the lung at autopsy of dust mantles and accompanying focal emphysema are the characteristic, diagnostic features of CWP. The focal emphysema is not visible on a chest radiograph. However, when the coal mantles become sufficiently numerous throughout the lungs, they produce the characteristic opacities on the chest

[66] Merchant, Kilburn, O'Fallon, Hamilton, & Lumsden, *Byssinosis and Chronic Bronchitis Among Cotton Textile Workers*, 76 ANN. INT. MED. 423 (1972).

[67] Sluis-Cremer, Walters, & Sichel, *Chronic Bronchitis in Miners and Nonminers: An Epidemiological Survey of a Community in the Gold-mining Area in the Transvaal*, 24 BRIT. J. INDUS. MED. 1 (1967).

[68] Kalacic, *Ventilatory Lung Function in Cement Workers*, 26 ARCH. ENVT'L HEALTH 84 (1973).

[69] Kibelstis, *supra* note 62; Hankinson, Reger & Morgan, *Maximal Expiratory Flows in Coal Miners*, 116 AM. REV. RESP. DIS. 175 (1977).

[70] Kleinerman, *supra* note 25.

radiograph. Focal emphysema accompanying coal mantles involves the central portion of the terminal lung unit including the three orders of respiratory bronchioles. In contrast to centrilobular emphysema, described below, focal emphysema causes minimal disruption of alveolar walls, no inflammation,[71] and produces almost no noticeable alteration in lung function.

Centrilobular emphysema is not restricted to those having been exposed to dust and therefore represents the form most commonly seen in the general population. This form of emphysema, while it involves the respiratory bronchioles leading into the terminal lung units, preferentially attacks the second and third order broncioles. This is in contrast to focal emphysema which tends to involve the first and second order bronchioles. Both lesions are seen near the center of the terminal lung unit, hence the term centrilobular emphysema. The characteristic effects of centrilobular emphysema include both a fairly extensive destruction of the adjacent alveolar tissue and the presence of chronic inflammation of the bronchiolar walls.[72] The destruction of alveolar tissue causes the smaller intrapulmonary airways to collapse during forced expiration, resulting in widespread airway obstruction. Furthermore, this tissue destruction decreases both the surface area as well as the blood supply available for gas exchange, resulting in abnormal gas distribution and diffusion. Thus in both its moderate and severe forms, centrilobular emphysema produces significant pulmonary functional impairment and disability.

While there is one study to the contrary,[73] no reliable evidence exists which would indicate a relationship between either simple CWP or coal mining itself and the incidence of centrilobular emphysema. The findings of the one study which did indicate such a relationship have been justifiably attacked on several

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[71] Heppleston, *Lung Architecture in Emphysema*, in FORM AND FUNCTION 6 (G. Cumming & H. Hunt eds. 1968) [hereinafter cited as Heppleston].

[72] *Id.*

[73] Ryder, Lyons, Campbell, & Gough, *Emphysema in Coal Workers' Pneumoconiosis*, 3 BRIT. MED. J. 481 (1970). This study claimed to have found a relationship between a decreasing ventilatory function during life and the amount of emphysema detected by point counting at autopsy, the lungs of 247 decreased miners. Further, the study demonstrated that the p type of CWP was also associated with a decreased ventilatory function.

grounds.[74] Cigarette smoking, on the other hand, is clearly a factor which contributes to the occurrence of centrilobular emphysema.

In panlobular emphysema, the most serious type, the airspace enlargement and accompanying tissue destruction often involve virtually the entire lobule (terminal lung unit) from the respiratory bronchioles to the most distal alveoli (air sacs). This kind of emphysema is relatively uncommon, sometimes occurring in persons, and to a lesser extent in the families of persons, who are deficient in an enzyme known as alpha-1-antitrypsin.[75] There is no evidence that this type of emphysema is related to coal mining or dust exposure.

Bullae, which are air containing spaces large enough to overrun or compress adjacent terminal lung units, may occur in association with widespread emphysema (bullous emphysema), or as an isolated lesion along the edges of an otherwise normal lung.[76] They may communicate at different levels of the bronchial tree, sometimes freely and other times through a partially narrowed connecting airway that may act as a one-way valve. Bullae exert their deleterious effects on lung function primarily by compression of adjacent lung tissue. Thus the size of the bulla and the rapidity of its expansion are important factors in determining functional impairment. The principle hazard from bullae lining the outer surface of an otherwise normal lung is the risk of spontaneous rupture, leading to collapse of the underlying lung.

Bullae may occur, in conjunction with the development of complicated CWP (PMF), immediately adjacent to the large opacities. They may result from tractional forces applied to the lung by the mass of scar tissue as it condenses and contracts in the process of forming the complicated lesion. The occurrence of

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[74] Gilson & Oldham, *Coalworkers' Pneumoconiosis*, 4 BRIT. MED. J. 305 (1970). The study was criticized on several grounds, including that it (the study) represented a biased sample of miners since many were seeking compensation, and that it included an appreciable number of subjects with PMF, which itself leads to a reduction in ventilatory function. Thus, the association between decrement in ventilatory function and emphysema was partially spurious.

[75] Hutchinson, Cook, Barter, Harris, & Hugh-Jones, *Pulmonary Emphysema and Alpha-1-Antitrypsin Deficiency*, 1 BRIT. MED. J. 689 (1971).

[76] Heppleston, *supra* note 71.

bullae in miners without complicated CWP are most likely not a consequence of occupational exposure.

Lung Cancer

Mortality figures indicate that British coal miners have had lower than expected death rates from lung cancer than non-miners.[77] Several studies suggest that this might not be the case for coal miners in the United States.[78] These divergent conclusions prompted a follow up study[79] on a group of Appalachian coal miners who had participated in a prevalence study in 1962-1963 to determine their mortality experience over the ten-year period from 1962 until 1972. The sample consisted of 3726 men who were initially identified according to chest radiograph, occupation, respiratory symptoms, smoking history, and ventilatory function testing.

Death certificates for all of the 451 men who died during the ten-year period were obtained and noted specifically was the number whose cause of death was listed as lung cancer. The study calculated the expected number of deaths due to lung cancer, using all males in the United States in 1968 as the reference population. The death rate for lung cancer in the miners was expressed as a standardized mortality ratio (SMR), which is the ratio of observed deaths to expected deaths times 100. Using this technique, the authors found an SMR for lung cancer in miners of 67, indicating that miners had a significantly lower death rate from lung cancer than the average of all males in the general population. This value was quite comparable to the British rates reported earlier.[80] It should be noted that twenty-one of the twenty-four miners dying from lung cancer were either current or former smokers, and smoking is an important factor associated with lung cancer.

It thus appears that neither coal workers' pneumoconiosis nor coal mining per se are causally related to the development

[77] Liddell, *Mortality of British Coal Miners in 1961*, 30 BRIT. J. INDUS. MED. 15 (1973) [hereinafter cited as Liddell].

[78] Scarano, Fadali, & Lamole, *Carcinoma of the Lung and Anthra-cosilicosis*, 62 CHEST 251 (1972); Enterline, *A Review of Mortality Data for American Coal Miners*, 200 ANN. NEW YORK ACAD. SCI. 260 (1972).

[79] Costello, Ortmeyer, & Morgan, *Mortality From Lung Cancer in U.S. Coal Miners*, 64 AM. J. PUB. HEALTH 222 (1974).

[80] Liddell, *supra* note 77.

of lung cancer. The occurrence of lung cancer in a coal miner must therefore be considered as a naturally occurring condition not related to the miner's occupation.

Asthma

Asthma is a disease characterized by an increased responsiveness of the trachea and bronchi to various stimuli. It is manifested by a widespread narrowing of the airways that changes in severity either spontaneously or as a result of therapy.[81] The cause of asthma in some instances appears to be clearly related to an altered immunological state (atopy, allergy) which is inherited. In other instances, however, the underlying cause for the asthma cannot be determined (intrinsic, non-atopic).

Coal miners as well as members of the general population are likely to inherit the predisposition (atopy) to develop allergic asthma as well as develop the non-allergic or intrinsic form. Asthmatic attacks can be triggered in the susceptible individual by a number of agents including infections, allergens, stimulation of airway reflexes, emotional factors, exercise, and physical and chemical agents including a wide variety of dusts, fumes and vapors. The fact that a miner develops bronchospasm upon exposure to the coal mine atmosphere does not mean the miner is allergic to coal dust. There is no evidence that coal dust is capable of acting as a true allergen. On the other hand, one study[82] has shown that large concentrations of inert dusts, including coal, can induce a mild bronchoconstriction in normal subjects and there is good reason to believe that subjects with hyperreactive airways should respond similarly to much lower concentrations of coal dust.

Asthma in a coal miner can be differentiated from other causes of airway obstruction that are persistent, and possibly related to occupational exposure, on the basis of history and also by a demonstration that the airway obstruction is responsive to medication as shown on ventilatory function tests.

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[81] American Thoracic Society, *Chronic Bronchitis, Asthma, and Pulmonary Emphysema*, 85 AM. REV. RESP. DIS. 762 (1962).

[82] McDermott, *Acute Respiratory Effects of the Inhalation of Coal-dust Particles*, 162 J. PHYSIOL. 530 (1962).

Heart Disease

Coal miners being evaluated for impairment of lung function as a consequence of their occupational exposure are generally in the age group of fifty-five to seventy-five years. This is also the age at which the prevalence of heart disease is fairly high in males. Because heart disease produces many symptoms which are not easily distinguished from those produced by lung disease, it is necessary to briefly enumerate the signs and symptoms of heart disease, indicating where confusion may arise.

Left-sided congestive (backward) heart failure is most commonly a result of left ventricular infarction (scarring following a heart attack), hypertension and disease of the aortic or mitral valve. This kind of heart failure results in pulmonary congestion. The symptoms of pulmonary congestion are exertional dyspnea (shortness of breath), orthopnea (breathless when lying flat), and paroxysmal nocturnal dyspnea (breathlessness or suffocation at night only relieved by sitting upright with the legs dependent or standing).

Differentiating between cardiac and pulmonary dyspnea is not always easy. Pulmonary dyspnea is usually associated with sputum production or wheezing, but at times cardiac dyspnea also manifests itself as wheezing. Pulmonary function testing helps differentiate obstructive airway disease, which produces an obstructive pattern, from cardiac failure, which produces a restrictive pattern. Physical findings such as hypertension, ventricular gallop sounds, murmurs of aortic or mitral valve disease and the electrocardiographic signs of old myocardial infarctions (heart attacks) and ventricular hypertrophy (enlargement) or strain, are confirmatory of a cardiac origin for the dyspnea (breathlessness).

When the left ventricle fails, symptoms secondary to pulmonary congestion, as described above, predominate. With time, the right ventricle also begins to fail. When a patient with left ventricle failure develops right ventricular failure, the more severe forms of dyspnea tend to diminish in frequency and intensity because the inability of the right ventricle to increase its output prevents the temporary imbalance between the two ventricles that had before led to the pulmonary vascular congestion.[83]

[83] E. BRAUNWALD, CLINICAL MANIFESTATIONS OF HEART FAILURE IN HEART DISEASE: A TEXTBOOK OF CARDIOVASCULAR MEDICINE 453 (1980).

Patients with right-sided heart failure develop enlargement and congestion of the liver, edema (fluid) in the dependent parts (feet and legs), ascites (fluid in the abdomen) and hydrothorax (fluid inside the rib cage but outside the lungs).

Cor pulmonale is a special type of heart disease and is secondary to extensive pulmonary disease. As indicated earlier, it is a sequel to complicated CWP in coal miners. It is also a sequel to a number of naturally occurring lung diseases in their advanced stages and when the pulmonary vascular bed has been extensively destroyed by either emphysema or a dense scarring process. Cor pulmonale starts as a recognizable strain on the right ventricle and proceeds to right ventricular failure with the manifestations described above.

CONCLUSION

Coalworkers' pneumoconiosis is a medically diagnosable condition resulting directly from the inhalation and deposition of coal dust within the gas exchange parts of the lungs. CWP exists in two forms—simple and complicated, based upon the characteristic lung reaction detected during life by the chest radiograph. Simple CWP seldom elicits symptoms or signs of respiratory impairment, while complicated CWP often leads to the development of shortness of breath, cough, and sputum production.

Simple CWP may lead to slight air trapping, mild obstruction within the airways, and slight alterations in blood oxygen tensions. Complicated CWP usually demonstrates moderate to marked degrees of restriction, obstruction of ventilatory function, reduction in arterial oxygen tensions, and pulmonary hypertension leading to cor pulmonale.

Coal miners are also subject to the development of naturally occurring diseases such as asthma, chronic bronchitis, emphysema, lung cancer, and the primary diseases of the heart. Chronic bronchitis, a disease of the airways, occurs frequently among coal miners, especially when compared to non-dust exposed populations. This "industrial bronchitis" is no different from the naturally occurring variety, at least with regard to its signs and symptoms. Industrial bronchitis does contribute a small amount of airway resistence, which probably accounts for the minimally detectable obstructive impairment seen in non-

smoking coal miners with either negative chest radiographs or those showing simple CWP.

The lungs and the heart are intimately associated and can respond in only a few very stereotyped ways to diseases that have a wide variety of origins. Careful inquiry and close examination, therefore, are necessary to determine the true cause of the symptoms. Thus, not every miner who is short of breath is necessarily suffering from CWP or any other respiratory disease that is a consequence of his occupation.

APPENDIX

ARGUMENTS OFTEN ASSERTED BY BLACK LUNG CLAIMANTS ON MEDICALLY RELATED ISSUES AND THE DEFENSE ATTORNEY'S MOST LIKELY RESPONSE

Issue 1: *Lack of Sensitivity of the Chest Radiograph*

Claimant's Argument

This claim is used when the autopsy of deceased miners, whose chest radiographs were classified as negative for CWP during life, indicates the presence of nodules on the lungs consistent with the early stages of CWP. This argument is furthered by the fact that chest radiographs generally do not detect rounded nodules within the lung gas exchange areas unless the nodules measure at least 3 mm. Thus, the claimant's attorney argues that the deceased miner suffered from black lung and should be compensated.

Defense Argument

The defense will likely argue, in response to the above, that since the chest radiograph is the best available diagnostic, semiquantitative tool available during life to detect the presence and severity of CWP, its results should be dispositive of the issue. While autopsy studies have shown that there may be a few widely scattered lesions typical of CWP in the lungs of deceased coal miners, who had negative chest radiographs, the defense will assert that it takes approximately three to four grams of coal dust in one lung before it shows up on the chest radiograph. Further, since studies of non-smoking miners with negative chest radiographs have shown that these scattered lesions have produced little or no functional impairment and are of no real consequence other than to demonstrate exposure to dust, compensation is not merited.

Issue 2: *Influence of Radiographic Technique*

Claimant's Argument

The claimant's attorney may argue that an overexposed

chest radiograph will almost invariably be under-read or classified lower than would a radiograph of the same subject taken under ideal exposure circumstances. Over-exposing a chest radiograph "burns out" the detail of the peripheral parts of the lungs where the pneumoconiotic opacities or nodules are best evaluated by the reader.

Defense Argument

The defense attorney will often argue that underexposed films will almost invariably be over-read and classified higher for CWP than would a radiograph of the same subject taken under ideal exposure circumstances. The underexposed film accentuates all of the "normal" bronchovascular and lung markings. Also, large amounts of muscle or fat in the chest wall cause some "addition" opacities of shadows to be cast on the film plate in such a way as to overlie the lung tissue. These "addition" opacities may be misinterpreted by the inexperienced reader as pneumoconiosis or as pleural thickening.

Issue 3: *Radiograph Interpreter Variability*

Claimant's Argument

In attempting to gain a liberal review from the hearing examiner of the claimant's medical evidence, the claimant's attorney will often seize upon the inherent variability of radiographic interpreters. An individual reader presented with the same chest radiograph on several different occasions will not always place the radiograph into the exact same category on each occasion. There is, in other words, an inherent "intra-reader" variation in classifying chest radiographs for pneumoconiosis. The claimant will further argue that when two or more different readers interpret and classify chest radiographs, the "inter-reader" variation is greater than that for the "intra-reader" variation.

Defense Argument

Defense attorneys will argue that the intra- and inter-observer variation is not necessarily excessive. Well-trained and experienced readers are generally able to keep the intra-observer variation to within 1/12 of the 12-point ILO U/C scale. Two well-trained and experienced readers will generally not

vary from each other's reading of the same film by more than 2/12 of the 12 point scale. The whole purpose of the National Institute of Occupational Safety and Health program for certifying proficiency by examination of B readers is to keep the variability of reading chest radiographs to a minimum. Physicians who qualify as B readers and who consistently use standard ILO U/C films when reading for pneumoconiosis are consistent in their readings. Inexperienced readers or those who do not compare unknown films with the standards produce much more variable interpretations.

Issue 4: *Medical Diagnosis of CWP Too Narrow*

Claimant's Argument

The claimant's attorney bases his argument on this issue upon the amendments to the Coal Mine Health and Safety Act of 1969. In determining the presence of CWP, these amendments have apparently swept aside many of the areas of uncertainty or disagreement within the medical community with regard to both the necessity of demonstrating typical radiographic findings and the establishment of an appropriately long exposure to coal mine dust. The claim is also based upon the fact that certain respiratory symptoms, such as cough and phlegm, while arising as a consequence of cigarette smoking or other naturally occurring factors, may also be a consequence of dust exposure ("industrial bronchitis") and may occur without producing the typical manifestations of CWP on the chest radiograph. Inherent in this argument is the assumption that all respiratory symptoms or pulmonary function changes in a miner are a consequence of his occupational exposure.

Defense Argument

The defense will argue, on the other hand, that the standard medical diagnosis of CWP, including a history of exposure to coal mine dust and the detection of the lungs' reaction to that dust by characteristic radiographic apearances, is one that has been accepted worldwide. Removing the chest radiographic criteria as a prerequisite for diagnosis of CWP during life opens the door for the compensation of a wide variety of respiratory and cardiac diseases that, while they may have respiratory symptoms, have only tenuous association with occupational ex-

posure in coal miners. Symptoms and pulmonary functional alterations are nonspecific and must be either related to the classical radiographic manifestations directly, or shown by well-designed epidemiological studies to occur in coal miners more often than in the general population, befor they are accepted as caused by occupational exposure.

Issue 5: *Aggravation of Pre-existing Respiratory Disease or Impairment*

Claimant's Argument

The claimant's attorney, in addressing this issue, will again rely upon the amendments to the Coal Mine Health and Safety Act of 1969. These amendments redefine coalworkers' pneumoconiosis to include not only any chronic respiratory disease that arises out of exposure to coal mine dust exposure, but any chronic respiratory disease or impairment that was "aggravated" by such exposure. The area of aggravation in medicine is quite broad. Thus, asthma, a naturally occurring disease of the airways, can be triggered and worsened by exposure to dusts, fumes, and gases encountered in the workplace. This argument is also based upon the well-known fact that persons with silica exposure, such as roof drillers, motormen, drillers into rock at surface mines, and underground shaft drillers, are at increased risk of contracting tuberculosis and when established in persons with silica exposure, the disease is more difficult to eradicate, even with effective antibiotic therapy.

Defense Argument

The defense will point out the difficulty encountered in trying to identify one component of a multifactoral disease causation scheme. The lungs are subjected to a wide variety of insults during life. For example, one inherits certain traits which might include the condition of atopy, the predisposition to develop allergies to various inhalants, to which the subject may on subsequent exposures manifest asthma. The lungs are insulted by viral and bacterial infections during their growth phase and may be damaged in such a way as to render them more susceptible to further injury later in life. A significantly large number of persons smoke cigarettes over a period of time sufficient to permanently damage the airways or cause destructive changes in the gas exchange areas (emphysema). These changes character-

istically do not manifest themselves by symptoms or functional decrements until about age fifty to fifty-five. Studies of the natural history of chronic obstructive airways disease show that many subjects continue to lose lung function at a rate that is two or three times faster than normal. Given these findings it may be difficult to prove that an occupational factor is causing aggravation of a naturally occurring or nonoccupational disease.

Issue 6: *Non-Specificity of Radiographic Opacities*

Claimant's Argument

The claimant's attorney will argue that the small, rounded opacities that are classified on the ILO U/C System are not specific to CWP. Certain other conditions such as sarcoidosis, miliary tuberculosis, and acute reinfection histoplasmosis, can produce rounded nodules that are indistinguishable from the nodules of CWP on the chest radiograph. Thus, the claimant's attorney will argue that where the worker's occupational history shows substantial coal mine employment, the rounded opacities should be presumed to indicate the presence of CWP.

Defense Argument

The defense will argue that naturally occurring diseases such as pulmonary sarcoidosis, miliary tuberculosis and acute reinfection histoplasmosis do produce rounded nodules on the chest radiograph that may be indistinguishable from CWP. The argument can be made that in the absence of a sufficiently long occupational exposure or the absence of an earlier chest radiograph demonstrating the nodules to have been present previously, the present nodules may be due to recently acquired naturally occurring disease and not due to pneumoconiosis. This argument is especially relevant if earlier chest radiographs are available and they do not demonstrate the changes of pneumoconiosis.

Issue 7: *Individual Susceptibility*

Claimant's Argument

Because individuals clear dust that has been deposited in the respiratory tract at differing rates, claimant's attorneys often argue that this phenomonon explains the rapid appearance or progression of the manifestations of CWP in a miner. There

are both rapid clearers and slow clearers. Given the same exposure, in terms of dust concentration, the slow clearer will retain more dust than the rapid clearer. Thus, those susceptible to increased dust retention will exhibit a more rapid appearance of the radiographic changes of pneumoconiosis than usual.

Defense Argument

The defense of this claim may utilize the arguments listed above (Issue 6) that the chest radiographic appearances of pneumoconiosis are non-specific. Thus, the more rapid the appearance or the more rapid the progression of nodular changes than usually expected given the occupational setting, the more likely the diagnosis of pneumoconiosis is in doubt. Furthermore, at this time, there are no reliable clinical diagnostic techniques, of easy and broad application, for determining whether a subject is a rapid or slow clearer of dust.

Issue 8: *Complicated CWP Misinterpreted*

Claimant's Argument

Large (greater than 1 cm.) nodules may be misinterpreted as tumors or as scarring due to tuberculosis or other inflammatory lesions. Hence, claimant's attorneys often must argue that such large nodules should be diagnosed as complicated CWP, rather than as a tumor or as scar tissue. The claimant's argument is bolstered by a showing that these nodules have arisen on a background of category 2 or 3 small nodules, are bilateral (occurring in both lungs), and are in either the posterior parts of the upper or the superior parts of the lower lobes.

Defense Argument

This argument can also be used in the defense of CWP claims. Coal miners may contract tuberuclosis or pneumonias which result in ill-defined, dense, scarred areas in the posterior segments of the upper zones of the lungs. Similarly, coal miners, especially those who smoke cigarettes, develop lung tumors in the same way as non-coal miners. Miners whose chest radiographs demonstrate large rounded or ill-defined densities on a background of category one or less should be considered to be suffering from a tumor or other non-occupational disease pro-

cess. It is most unusual to develop PMF or complicated CWP on a background of less than category two or category three simple CWP.

Issue 9: *Partitioning the Effects of Occupation and Cigarette Smoking*

Claimant's Argument

The claimant's attorney will argue that it is not medically feasible to separate the effects upon lung function of the relative contributions of occupational exposure and cigarette smoking within an individual because the partitioning is based upon epidemiological (population) studies. The results obtained in such studes are applicable to groups but not to individuals. The amendments to the Black Lung Act also require that medical testimony be applied to a specific individual if attempts are made to partition respiratory functional effects according to multiple causes.

Defense Argument

Defense attorney's will respond by arguing that in the medical field, the use of epidemiological studies of populations permits the identification and quantification of individual factors contributing to diseases or manifestations (symptoms, function) that have multifactoral origins. While data derived from such studies may not be directly applicable to an individual subject, they do provide ranges over which one may estimate the relative contributions of various components to the multifactoral disease or impairment. Such studies give an indication of the likelihood that one factor or the other will produce all, part, or almost none of the disease manifestations. For example, by studying smoking and non-smoking coal miners exposed to coal mine dust, and smoking and non-smoking control subjects not exposed to coal mine dust, it is possible to determine, on average, the relative contributions of smoking and coal dust exposure to respiratory symptoms, function, and premature death. Most medical diagnoses and judgements are based upon probabilities or likelihoods that an agent or agents caused a certain manifestation (symptom, decrement in function, death). These judgements are based partly on personal experience, replicated over years of practice, but more reliably, upon shared observa-

tion with other colleagues and knowledge of the results of well-designed epidemiological studies.

Issue 10: *Relationship of Autopsy Findings to Living Miner's Functional Status*

Claimant's Argument

The claimant's attorney may argue that the functional status of an individual miner, during life, cannot be determined by relying solely upon a pathologist's examination of the lungs or of microscopic sections of the lungs at autopsy. The pathologist can detect and quantify the characteristic changes that establish the diagnosis of CWP. If the airways are also examined, the pathologist can make a diagnosis of chronic bronchitis. Functional impairments during life, however, may not produce detectable alterations at autopsy examination.

Defense Argument

The defense attorney's argument will be that pathologists have been for many years inferring from the examination of structural alterations of the lung at autopsy whether a given process is sufficiently widespread as to cause not only functional impairment, but also the death of the individual. In making sections of the lungs for microscopic examination, the pathologist, through training, is taught to preserve the most representative and most diseased tissues. Thus, the finding of a few relatively early lesions of simple CWP within a large field of normal lung tissue showing little or no disruption of the alveolar wall, no appreciable thickening of the walls with collagen, and normal sized, delicate, pulmonary capillaries traversing the alveolar walls, permits the pathologist to state that these changes would not likely have interfered with lung function during life.